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Volume VIII — March 1957 — Number 1

CONTENTS

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EDITORIAL

PHYSICIAN-HOSPITAL RELATIONSHIPS

In November 1956, a joint declaration on physician-hospital relationships was ratified by the Iowa State Medical Association and the Iowa Hospital Association. It clearly delineates radiology and pathology as the practice of medicine and clearly delineates the place of hospital departments of radiology and pathology. It may be valuable to Canadians who are trying to define the role of radiologists and pathologists in proposed government health plans. It says in part:

The ownership and maintenance of the laboratory and X-ray facilities and the operation of same under this joint declaration is a proper function of a hospital.

Pathology and radiology services performed in hospitals are the product of the joint contribution of hospital, doctors and technicians, but these services constitute medical services which must be performed by or under the direction and supervision of a doctor and no hospital shall have the right, directly or indirectly, to direct, control, or interfere with the professional medical acts and duties of the doctor in charge of the pathology or radiology facilities or the technicians under his supervision.

The contract between the hospital and doctor in charge of the laboratory or X-ray facilities may contain a provision for compensation of each upon which they mutually agree, provided, however, that no contract shall be entered into which in any way creates the relationship of employer and employee between the hospital and the doctor.

The hospital bills shall properly include the charges of pathology and radiology services as long as the name of the doctor is stated and it clearly appears that the charge is for medical services.

Fees for radiology and pathology services must be paid for as medical and not hospital services. In all cases this requires payment by Blue Shield and not by Blue Cross.

D.L.M.

THE JOURNAL OF THE
CANADIAN ASSOCIATION OF RADIOLOGISTS

Volume VIII March 1957 Number 1

EVENTRATION OF THE DIAPHRAGM

A. TAMAS, M.D., and J. S. DUNBAR, M.D.
Montreal, Quebec

The term eventration of the diaphragm is an unsatisfactory one. It is derived from the Latin e — out, and venter — belly. This term has come to mean elevation of part or all of one leaf of the diaphragm.[18] It will be used in this communication in spite of its admitted inadequacy, because of its use since the condition was first recognized in the late 18th Century by Petit, and the lack of any other recognized terminology descriptive of the lesion.[3]

Elevation of part or all of the diaphragm can be due either to phrenic nerve palsy or to absent or deficient muscle fibres. It is not due to a dehiscence or actual defect of the diaphragm.

Total Eventration

When there is known post-traumatic phrenic palsy (as in phrenic-crush or avulsion for pulmonary tuberculosis) the resulting unilateral diaphragmatic paralysis causes upward displacement of the affected leaf and diminution or absence of its normal contraction. This is recognized by its position and by the diminution of its excursion under the fluoroscope. So-called "paradoxical movement" is an important sign. It was first described by Keinbock in 1898,[25] in connection with a case of pyopneumothorax, but has come to mean upward movement of the affected leaf of the diaphragm on inspiration while the normal leaf moves downward. It is most obvious on rapid inspiration (sniffing). When slight in degree it may be elicited only on rapid inspiration. Paradoxical movement has been pointed out repeatedly as a sign of complete paralysis of the diaphragm. It may, however, only indicate weakness of the diaphragm, when the affected leaf can be seen to descend slightly on a long, slow inspiration, but on a quick inspiration will ascend temporarily and then either stay up or descend, depending on the degree of neuro-muscular deficit.

Thus, when the etiological agent is clearly known or operatively produced, a high, weak or paralysed diaphragm may be referred to as a diaphragmatic or phrenico-diaphragmatic palsy or paralysis. When the etiology is unknown or when it occurs in newborn infants, where a phrenic nerve lesion may be suspected, but is difficult to prove, the abnormality is called "total eventration of the diaphragm".[21]

CASE REPORTS

Case 1

C. S. At birth, this female infant presented face to pubis and delivery was difficult. The infant was cyanotic for 15 minutes after birth and resuscitation was required with intra-tracheal intubation. Irregular respirations continued during the first two days of life. The child was admitted to the Montreal Children's Hospital at age 2 days. In hospital, she improved progressively and was discharged at age 2 weeks, with no cyanosis. The cyanosis never returned. At age 6 months, the child was taken into a foster home. The foster mother noted that she was "stiff all over" and had difficulty in moving her right arm. This condition showed some improvement. When last seen at age 3, the child had a mild right hemiplegia with the upper extremity involved more than the lower. She was doing well otherwise. There was equivocal mental retardation.

The postero-anterior radiograph, figure 1A, at age 2 days, shows marked elevation of the entire right leaf of the diaphragm with displacement of the mediastinum to the left and decreased aeration of the right lung. This elevation diminished in degree with growth. The final X-ray examination at age 3, figure 1B, shows moderate residual elevation of the right leaf of the diaphragm. It moved synchronously with the left leaf, but excursion was considerably reduced. No "paradoxical movement' was demonstrated, but because of the child's age, voluntary "sniffing" could not be elicited and therefore slight "paradoxical movement" may have been missed. There was no displacement of the mediastinum to the left, as there had been. Air exchange in the right lung under the fluoroscope was only slightly less than in the left.

Case 2

Baby S. P. Male infant, two weeks post-mature. The presentation was face to pubis, and labour was difficult. Birth weight was 8½ pounds. On admission to the Montreal Children's Hospital at age 1 day, the child had neck stiffness and head retraction. The face was bruised, there was swelling over the vertex with molding of the head, right subconjunctival hemorrhage and obvious right facial palsy. Spinal puncture showed xantho-chromic fluid with many red blood cells, but normal pressure. The postero-anterior radiograph (Fig. 2A) showed elevation of the right leaf of the diaphragm. Under the fluoroscope, the right leaf showed "paradoxical movement" and the heart shifted to the left on expiration. There was also exidence of right-sided bronchopneumonia. Treatment included oxygen, moisture and antibiotics. From age 4 days, there was progressive improvement. On the 9th day, fluoroscopy showed some elevation of the right leaf, but no "paradoxical movement". Air exchange

in the lungs was improved. Facial palsy was improving. The child was discharged at age 10 days, markedly improved with no dyspnoea or cyanosis.

Follow-up examination at age 2 months, Figure 2B, showed normal position and movement of the diaphragm and normal air exchange.

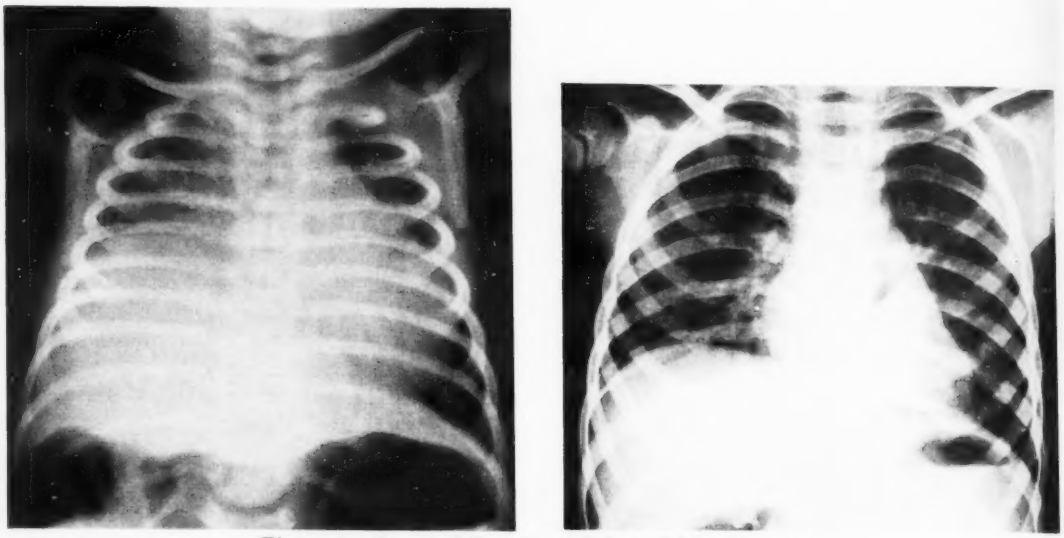


Figure 1. Case 1, S.C., (a) age 2 days. (b) age 3 years.

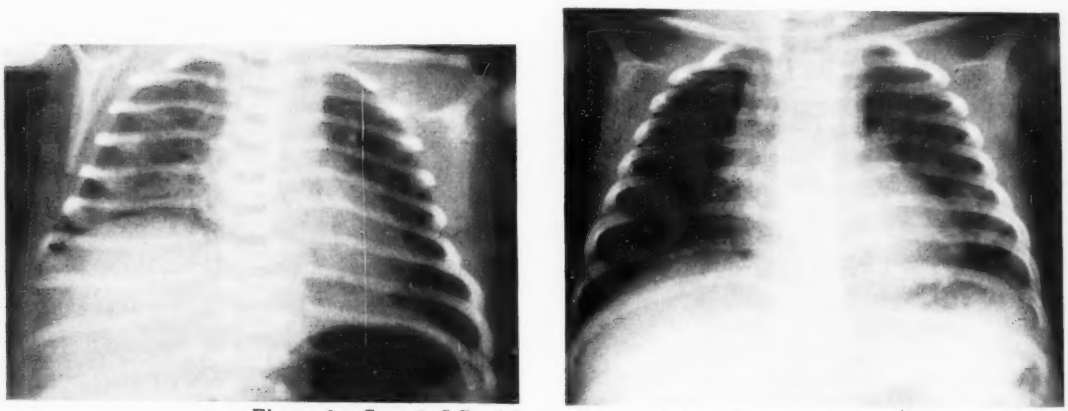


Figure 2. Case 2, S.P., (a) age 4 days. (b) age 2 months.

Case 3

Baby Girl A. N. Normal pregnancy and delivery. Cyanosis appeared immediately after birth and progressed to asphyxia livida. Artificial respiration was performed and spontaneous respiration recommenced. The infant was placed in an incubator and oxygen was given continuously. She remained dyspnoeic and became cyanosed when out of oxygen.

Radiological examination at age 3 days (Fig. 3) showed elevation and "paradoxical movement" of the entire right leaf of the diaphragm.

Physical examination did not reveal any neurologic signs. The respiratory distress was the only positive finding.

The baby failed to improve. On the 4th day of life, a right thoracotomy was performed. The right leaf of the diaphragm was seen to be thin and elevated. No localized lesion or defect was demonstrated in it. The phrenic nerve was identified, but was not stimulated or biopsied. The leaf of the diaphragm was plicated in two layers transversely from the chest wall to the mid-line. Post-operatively, the child did extremely well. The cyanosis and dyspnoea disappeared immediately after operation and never returned. Fluoroscopic examination of the chest a few days after operation showed that the right leaf of the diaphragm was in approximately normal position, but that its excursion was limited or absent. Antero-medially, the diaphragm still showed "paradoxical movement" with an upward bulge on inspiration. Postero-laterally, there was definite downward contraction synchonous with the left leaf, but of short excursion. The child was discharged at age 4 weeks. Postero-anterior films at age 5 weeks (Fig. 4a) showed the right leaf of the diaphragm only slightly elevated and with no localized defect on lateral projection. The final examination was made at age 8 months. At this time the child had no respiratory distress or cya-

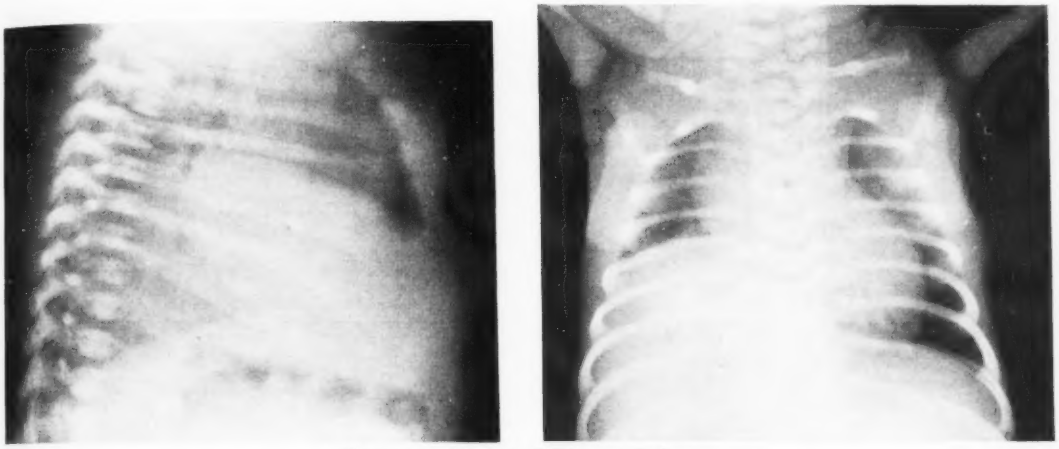


Figure 3. Case 3, A.N., age 3 days.

nosis. There has been no respiratory infections. She appeared quite healthy, though somewhat underdeveloped and underweight. Fluoroscopic examination (Fig. 4B) showed that the elevation of the right leaf of the diaphragm had increased since the examination one fonth after operation. It was now at least moderate in degree. It showed marked "paradoxical movement", particularly anteriorly. There was, however, slight downward movement of the entire diaphragm toward the end of inspiration. The mediastinum moved slightly to the left on inspiration.

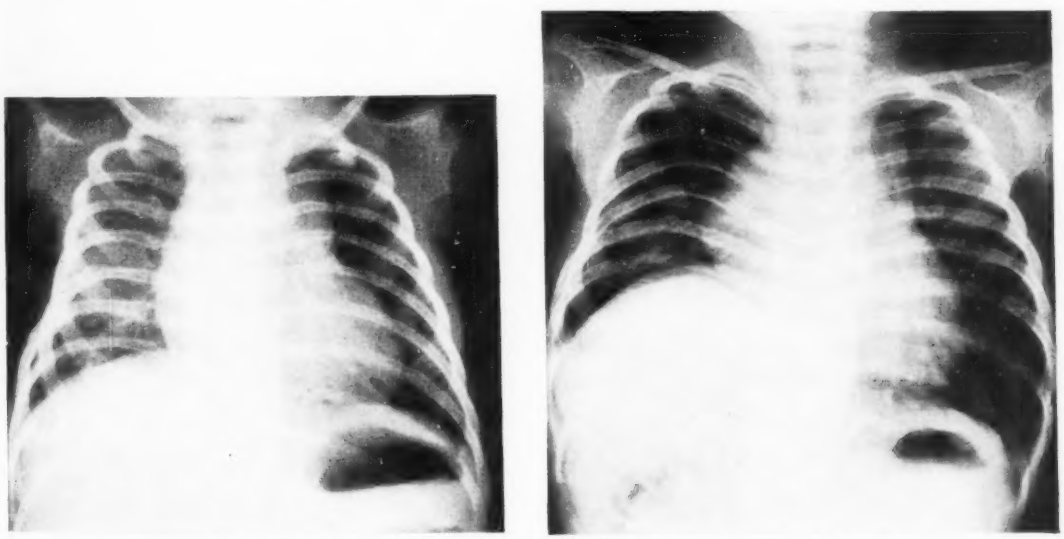


Figure 4. Case 3, A.N., (a) age 5 weeks. (b) Post-operative — age 8 months.

Partial Eventration

Part of a leaf of the diaphragm may be elevated and the remainder normal, both in position and muscular contraction. In these cases, it seems much less likely that neurologic lesions are responsible.

CASE REPORTS

Case 4

A. D. Female, age 6 months. Born by Caesarian section because the previous delivery had been Caesarian due to "hemorrhage at 8 months." Birth weight was 8 pounds, 13 ounces. The infant was perfectly well until one month before admission, when she developed a respiratory infection. She was admitted to another hospital where pneumonia was diagnosed and treated. During that admission, an abnormality was demonstrated in the right side of the chest. On discharge, re-admission for surgery was advised, and she was brought to the Montreal Children's Hospital. On admission, physical examination revealed no significant abnormality and in particular, no dyspnoea. Figure 5 shows marked upward bulging of the antero-medial portion of the right leaf of the diaphragm with slight

to moderate displacement of the heart to the left. On fluoroscopy, the entire leaf moved downward, on inspiration, but the abnormal portion less than the remainder. The bulge became more obvious on inspiration. Air exchange was approximately equal. Six days after admission, a right thoracotomy was done. The diaphragm was found to be thin and "tendinous" in the area of bulging and this area contained the right lobe of the liver. The abnormal portion of the diaphragm was partially removed and the defect repaired by overlapping the edges. The pathologist's report on the biopsy specimen was "tendinous diaphragmatic tissue with complete absence of muscular diaphragm".

The child did well post-operatively. Post-operative films after one week showed a normal contour of the right leaf of the diaphragm. She was followed until age 21 months, and during that time grew and developed normally.

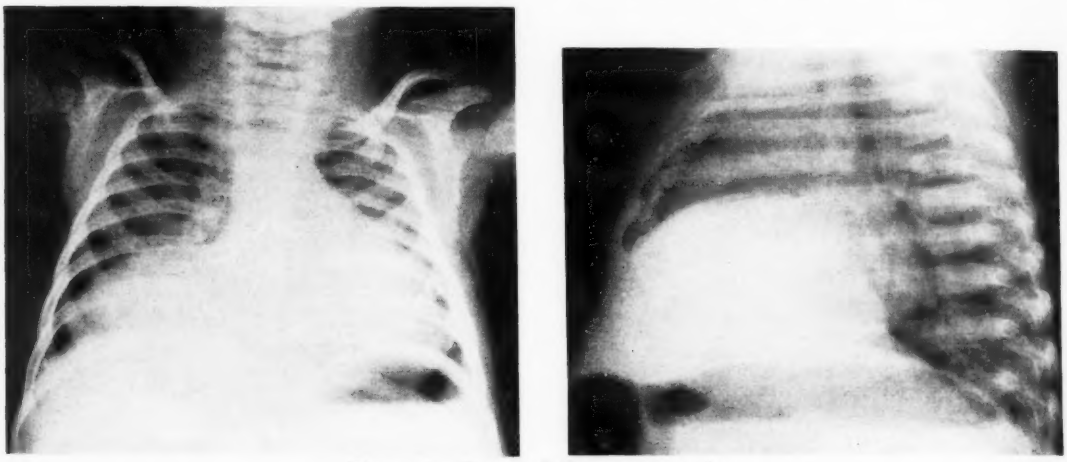


Figure 5. Case 4, A.D., age 6 months.

Case 5

D. D. Female, age 1½. The chest was first radiographed because of some suspicion that the child had "swallowed something" when the parents were absent. There were no abnormal findings on physical examination but radiologically an antero-medial bulge in the right leaf of the diaphragm was clearly demonstrated (Fig. 6). This child is an elder sibling of Case 4, A. D. For this reason, she was followed to age 5. The localized bulge of the right leaf did not change significantly during this time. She remained entirely asymptomatic.

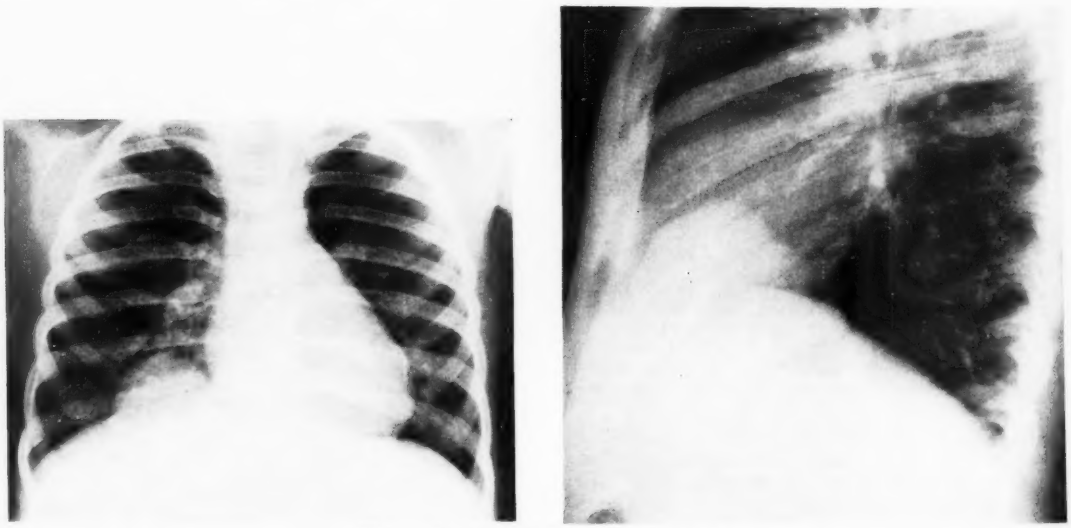


Figure 6. Case 5, D.D., age 4 years.

Case 6

M. D. The mother of Cases 4 and 5 above. Her chest was examined radiologically because of the finding of similar diaphragmatic contours in her two daughters. A slight, but definite bulge in the diaphragm antero-medially on the right side was present. She was obese, but entirely asymptomatic and had no history of any chest disease.

Case 7

P. C. male age 7 months. This child was an apparently normal delivery at home. He was well until age 5 months, when he was admitted to another hospital because of "pneumonia and gastro-enteritis". He was treated there for seven weeks. He was then transferred to the Montreal Children's Hospital for treatment of a "diaphragmatic hernia". found on X-ray examination.

On physical examination no abnormality was demonstrated. In particular, there was no evidence of any dyspnea or cyanosis.

Films made at age 6 months, at the other hospital, had shown marked elevation of the left leaf of the diaphragm in its anterior two-thirds (Fig. 7). This diaphragmatic bulge contained the stomach and probably the spleen. The heart was slightly, but definitely displaced to the right.

Ten days after admission, thoracotomy was done. A localized bulge in the left leaf of the diaphragm anteriorly was found. The thin diaphragm was partially resected. The specimen removed measured approximately 6.5 by 2 cms., and was oval in shape. The pathologist reported that "The only definite abnormality is the absence of muscle in large areas and the presence of muscle only in a peripheral rim at one end of the specimen. From the histological point of view, the dome of the 'defect' is tendinous tissue and therefore compatible with the tendinous portion of the diaphragm. This is presumably compatible with eventration".

Post-operatively, the child did well. Chest films one week after operation (Fig. 8) showed only a minimal residual bulge of the left leaf of the diaphragm antero-medially.

We have been unable to get any follow-up information on this child.

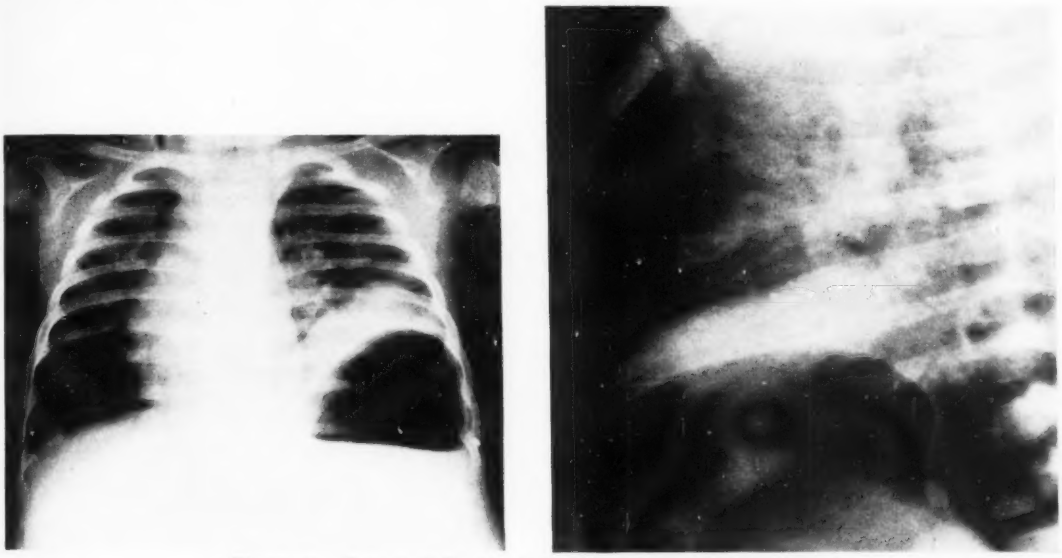


Figure 7. Case 7, P.C., age 5 months. Pre-operative films.

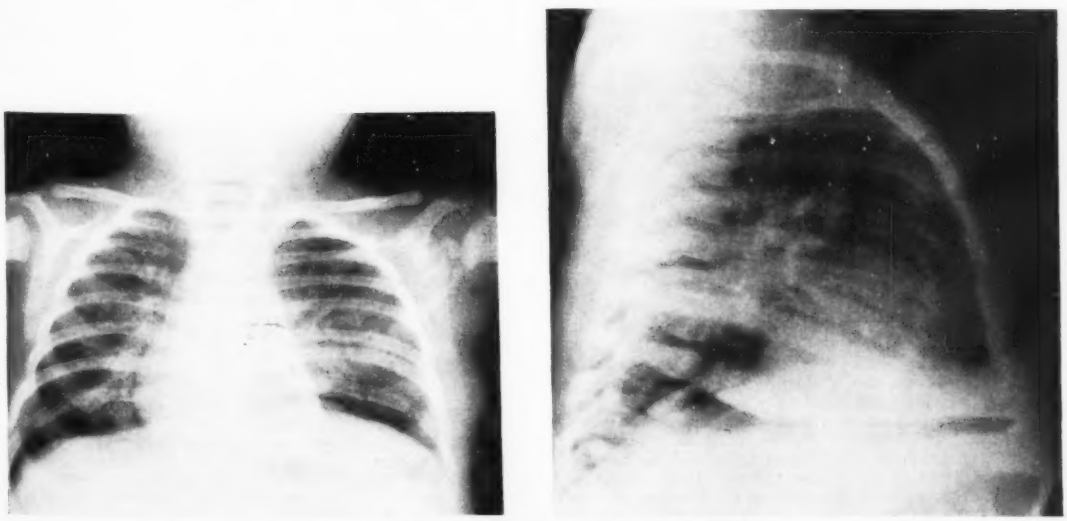


Figure 8. Case 7, P.C., 1 week post-operative.

Case 8

H. A. Male, 9 weeks. Normal birth and development until approximately age 8 weeks, when a respiratory infection necessitated admission to hospital. On admission, he had cough, fever, and vomiting. Clinical and radiological findings were consistent with pneumonia, but the radiological changes in the lungs were minimal. In addition, there was a well-marked elevation in the middle of the right leaf of the diaphragm (Fig. 9). On fluoroscopy, this moved less than the periphery of the right leaf. The heart shifted slightly to the left on expiration. Air exchange in the lungs was normal. The heart appeared normal. The periphery of the right leaf of the diaphragm moved synchonously with the left leaf. Progress in hospital on antibiotics was entirely satisfactory. He was discharged one week after admission, and seen a few days later in the Out-patients' Clinic. At this time, he was doing well with satisfactory recovery from pneumonia and satisfactory weight gain. Films at that time showed no change in the diaphragmatic abnormality. The patient was seen again in February, 1957, age 11 months. He was well-developed, and healthy, and had had no respiratory symptoms. Another X-ray examination showed no significant change in the diaphragm.

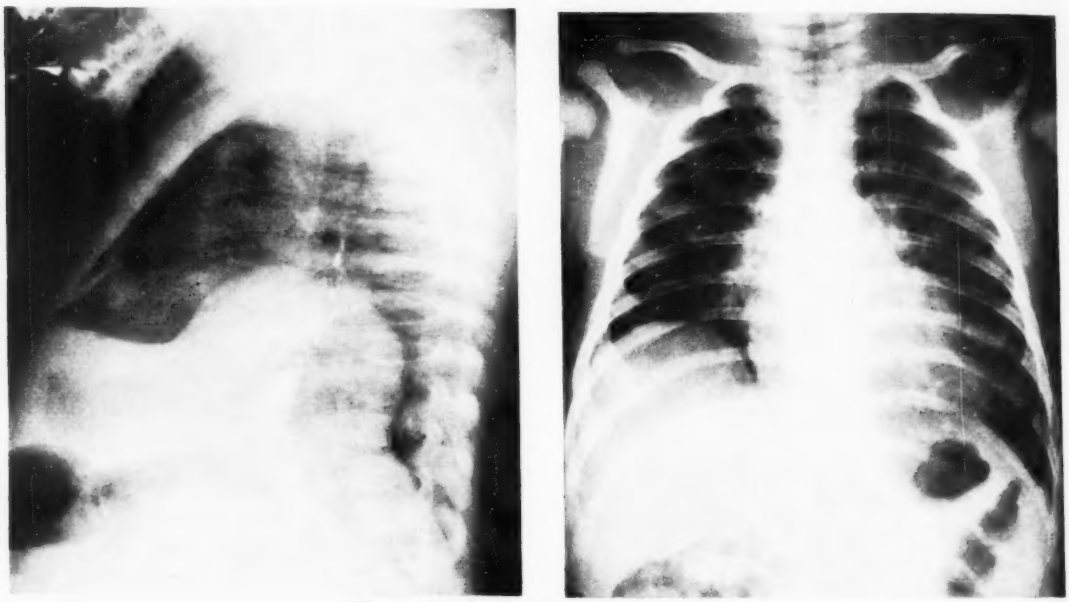


Figure 9. Case 8, H.A., age 9 weeks.

Case 9

Mrs. P. B. Age 54. She was an employee at the Montreal Children's Hospital and had had annual chest films made for at least six years (Fig. 10). They all showed fairly marked and well-localized elevation of the antero-medial one-third of the right leaf of the diaphragm. She had always enjoyed good health. Her case is of interest because of the question which has been raised from time to time, as to whether this type of diaphragmatic elevation and presumed weakness could give rise to difficulty during pregnancy or parturition. She has five children, and, by her own account, had had no difficulty with any of them. She said, in fact, that it was "a pleasure having each of them".

Case 10

M. K. This child was admitted to hospital at age 4 months for repair of a cleft palate. Chest films (Fig. 11) showed a well-localized elevation of the middle of the right leaf of the diaphragm. About one-half of the right leaf was involved. There were no chest signs or symptoms. The patient was discharged in good condition following repair of the cleft palate. He has been followed to age 3½ years and remains asymptomatic and is growing and developing normally. Further chest films show that the elevation of the middle of the right leaf of the diaphragm is becoming less marked. (Fig. 12.)

Case 11

S. C. Female, age 5½ months. Believed to be a premature infant of 7 months gestation, but this is uncertain. Pregnancy was uneventful. The child is said to have had a "stormy post-natal course with sclerema" in a premature nursery where she was kept until age 6 weeks. Birth weight was 4 pounds, 6 ounces. Following discharge, she gained weight at home, but was not considered healthy. At age 5 months, the weight was 10 pounds, and this was never exceeded. She was noted to tire easily with feeding and to become dyspnoeic with some cyanosis about the lips. At 5 months, vomiting and diarrhœa appeared, necessitating admission to hospital. On admission, physical examination revealed a small, poorly nourished, poorly-developed infant with good colour in oxygen but with cyanosis when out of oxygen. Her appearance was that of chronic illness. She was, however, active and alert. There was marked dorso-lumbar scoliosis convex to the left which was never explained. The liver was one fingerbreadth below the right costal margin. There was fever, and signs of bronchopneumonia were present.

Other findings were not relevant. In hospital she continued to do poorly and failed to gain weight. Cyanosis when out of oxygen continuel. In the premature nursery an inconstant murmur had been noted at the left sternal border in the 4th interspace, but this was not heard here.

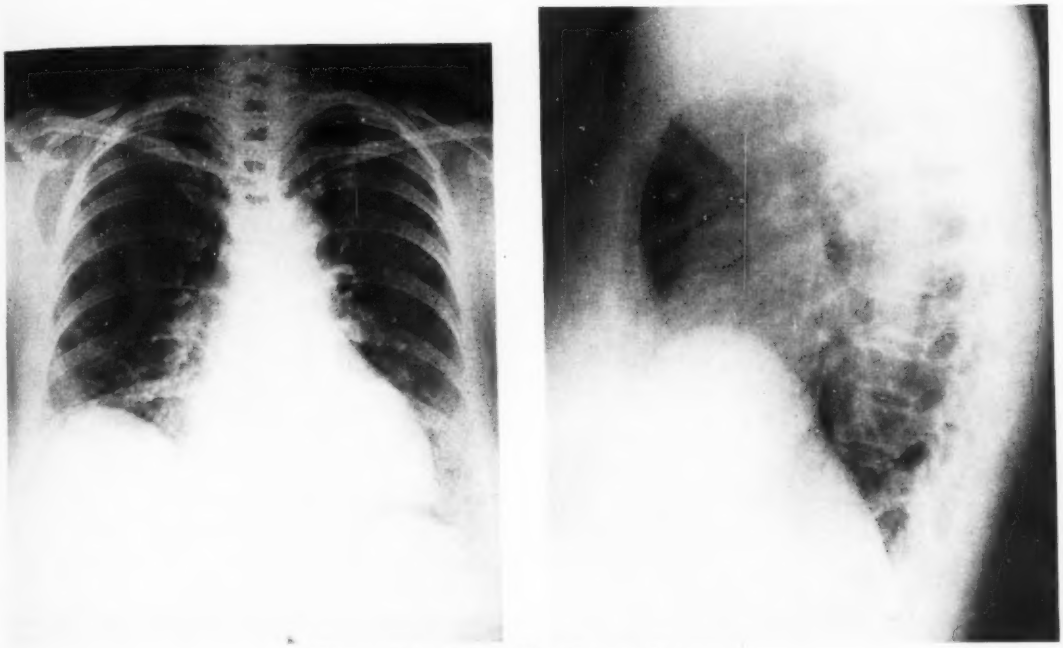


Figure 10. Case 9, Mrs. B., age 54 years.

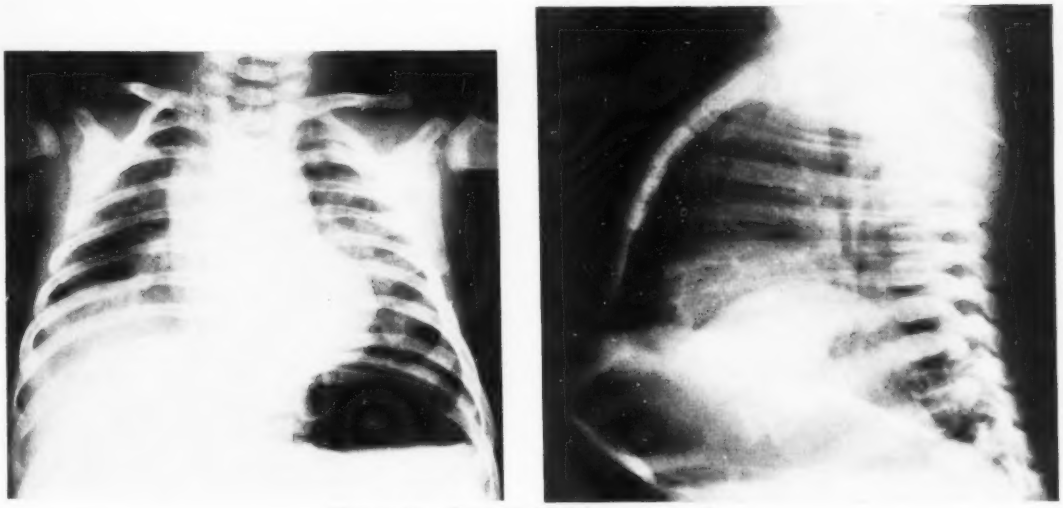


Figure 11. Case 10, M.K., age 4 months.

Electrocardiagrams showed right ventricular hypertrophy which was slightly but definitely above normal for her age. X-ray examination (Fig. 13) showed a marked elevation of the anterior two-thirds of the right leaf of the diaphragm which had a slightly lobulated appearance. The posterior margin was normal in position and in excursion.

The heart was difficult to assess because of the marked scoliosis and the abnormal right diaphragm which displaced it to the left. It was felt to be larger than average, but not necessarily abnormal in size or contour. Lung vascularity was perhaps slightly increased, but again difficult to assess. The left leaf of the diaphragm was normal in position and movement. When the child was out of oxygen, the oxygen saturation of the peripheral blood was approximately 80% at rest falling to about 65% on crying.

Because of the continuing failure to thrive and the cyanosis and the absence of clear indication that congenital heart disease was responsible for these findings, a thoracotomy for repair of the right diaphragmatic lesion was done at age 6 months. The elevated and thinned portion of the right diaphragmatic leaf was identified and was repaired by suturing intact diaphragm to costal margin and imbricating the excess portion. A biopsy was taken from the abnormal portion of the diaphragm.

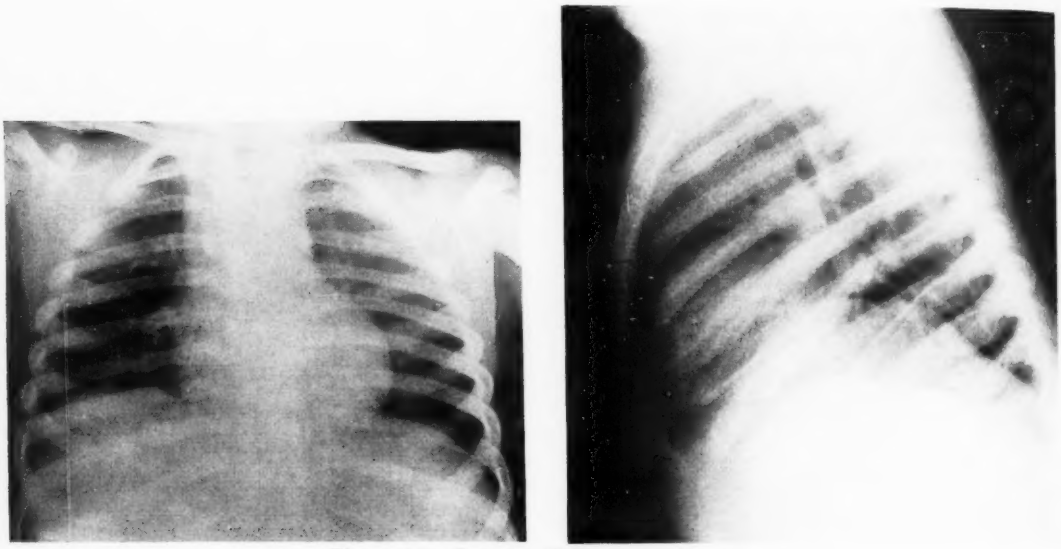


Figure 12. Case 10, M.K., age 2½ years.

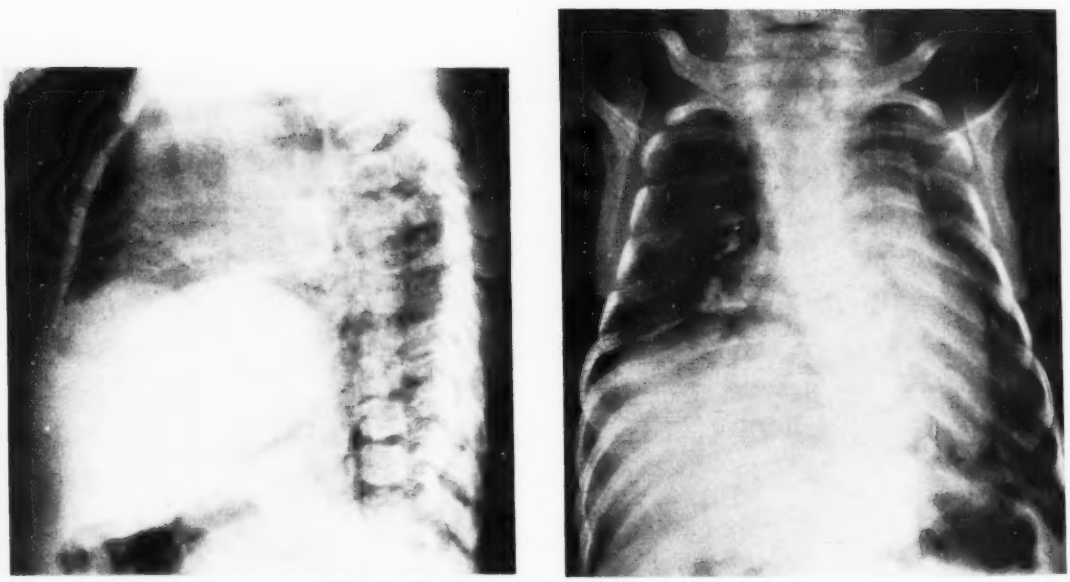


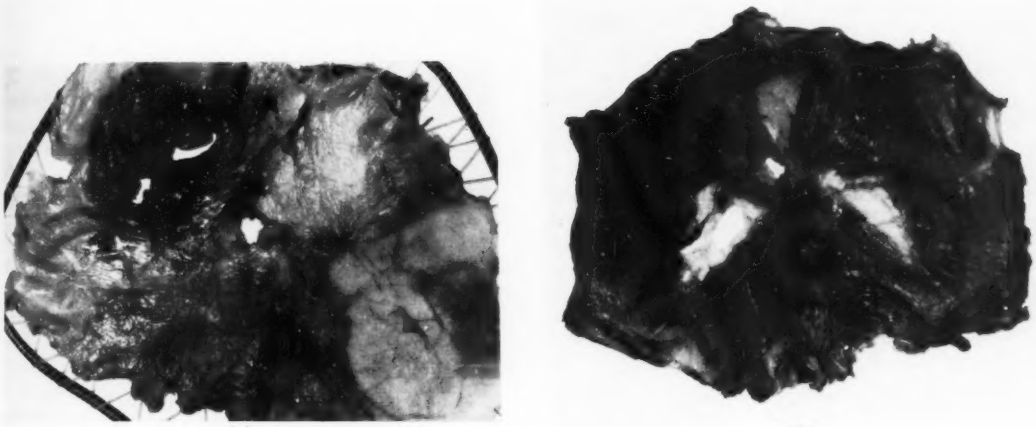
Figure 13. Case 11, S.C., age 5½ months.

The anaesthesia was continuously difficult, with a considerable amount of moisture in the lungs and in the trachea, which required repeated aspiration.

The child stood the procedure poorly. The immediate post-operative course was unsatisfactory and death occurred two hours after operation. The post-operative chest film showed good expansion of both lungs and relatively normal contour of the diaphragm and return of the heart approximately to the midline.

At autopsy, the repair of the diaphragmatic lesion was undone to try to re-establish the lesion as far as possible (Fig. 14A). The diaphragmatic specimen removed in toto showed that the absence of normal muscle tissue in the area which had been elevated pre-operatively was due to absence of muscle fibre, as far as could be determined from gross examination. Anatomically, the abnormality was bilateral.

The microscopic sections showed that muscle bundles were present, but scattered and separated by fibrous tissue instead of forming a relatively continuous sheet. There was a large uncovered (patent) foramen ovale with slight to moderate hypertrophy of the right ventricle. The lungs showed acute congestion with early pneumonia. There was equivocal evidence of pulmonary hypertension.



(a) (b)

Figure 14. Case 11, S.C., (a) Photograph of the superior surface of the diaphragm removed at autopsy. The pale areas represent tendinous tissue and the darker areas represent muscle. The difference is best seen at the lower right-hand corner where a thin layer of muscle fuses with a fibrous zone. The dark area in the upper left-hand corner is the site of operative repair. (b) The diaphragm from a 4 year-old child to illustrate the normal appearance of the diaphragm.

Discussion

There are four conditions which, though frequently quite distinct, form a series of related abnormalities. These are — physiologic variations in the normal diaphragmatic contour, diaphragmatic hernia, phrenico-diaphragmatic palsy, and eventration of the diaphragm.

1. Physiologic Variations in the Diaphragmatic Contour

European and particularly German authors have recognized for many years that there are marked variations in the supposedly smooth arc of the diaphragm, anatomically and radiologically, which can simulate organic disease. Singer and Boiken[23] first discussed these in the English language literature in 1933, and since then very little has been added. They drew attention to the now familiar "costal digitation" of the diaphragm seen in full inspiration, and therefore best seen in emphysema, and often simulating pleural adhesions.[20] They also noted an "antero-medial bulge" in the right leaf of the diaphragm. This is present in our case, M.D., the mother of the two children with more marked changes. It is a normal phenomenon, not infrequetly seen in routine X-rays. Eisler and Thomas, in anatomical studies reported in 1911, noted it. They believed that it was due to division of the right leaf of the diaphragm into a powerful postero-lateral portion and a much weaker antero-medial portion in normal individuals. The postero-lateral portion is composed of posterior lumbar fibres forming the crura and lateral costal portion from the 9th rib down. The antero-medial portion arises from the xyphoid process of the sternum, and the 7th and 8th ribs. These two portions may be divided by an actual groove in the normal diaphragm.

This antero-medial bulge may be seen at all ages and is, we believe, always asymptomatic. It is most marked in our Case 9, P.B., a healthy mother of five children.

2. Diaphragmatic Hernia

The term diaphragmatic hernia generally denotes a group of abnormalities which are fairly well recognized. They include the congenital types, which may be postero-lateral (Bochdalek), retro-sternal (Morgagni), hiatus, and sometimes central lesions, as well as traumatic hernia.[16,19] In infancy and again in adult life, hiatus hernia is a subject of considerable controversy, but this is not germane to the present discussion. The typical diaphragmatic hernia of infancy ir responsible for an alarming displacement of abdominal contents into the thorax, including almost any viscera except genitals, bladder and rectum, with marked mediastinal displacement and symptoms of acute respiratory distress. The term "diaphragmatic hernia", however, does not clearly define the condition of the diaphragm allowing this massive displacement of abdominal viscerae. Most surgeons include as abdomino-diaphragmatic herniae, cases with and without a sac. When there is no sac, there is an actual defect in the diaphragm. When a sac is present, it is a markedly thinned portion of the diaphragm enclosing the displaced abdominal organs in the thorax. Gross states[15] that the posterior

(Bochdalek) type is without a sac in 90% of cases, the hiatus always has a sac, and the anterior (Morgagni) has a sac in about 50% of cases. He further states that when a sac is present it limits the upward displacement and that the extreme displacements are not seen in those with a sac. It is evident that the distinction of diaphragmatic hernia with a well-formed sac from so-called partial eventration of the diaphragm is a matter of degree, or even of terminology, rather than essential.[5] The only justification for classifying the present cases as "partial eventrations" is that they tend to give a totally different clinical picture. In fact they do not usually produce symptoms and signs.

3. Phrenico-Diaphragmatic Palsy

There is a considerable number of cases, of which only a few are well studied and documented, of diaphragmatic paralysis in the newborn associated with dyspnea, cyanosis and at times, death. Newborn diaphragmatic palsy is usually right-sided, and is generally associated with signs of brachial plexus injury.[8] There are eleven previously recorded cases of "isolated diaphragmatic paralysis"[22] in which no evidence of brachial plexus injury has been found.[4] These include one or two in which there was fairly good indication of an isolated phrenic nerve injury. One of these was in a child whose neck was accidentally lacerated when the cord, tightly wound around the neck, was cut during delivery[8]. Other cases are assumed to be phrenico-diaphragmatic paralysis without brachial plexus injury and it is supposed that they may be due to such causes as pressure of forceps over the phrenic nerve in the neck. It has been suggested by Rupilius that intra-uterine position and pressure may cause an isolated phrenic nerve palsy, but this is at best an hypothesis.

It is interesting that authors reporting diaphragmatic paralysis seem to be unaware of the concept of eventration of the diaphragm and vice versa. It is in cases of total elevation of one leaf of the diaphragm, usually the right, assumed to be due to phrenic nerve injury, but not proven, that the two conditions become indistinguishable, both in published reports and in our Case 3. Even the cases who have died have generally not had sufficiently careful and convincing post-mortem study to make this differentiation. Our Case 1, with hemiplegia and marked right-sided diaphragmatic elevation, is clearly a paralytic lesion, and is the first recorded case of a birth injury followed by hemiplegia with this type of symptomatic diaphragmatic paralysis.

In diaphragmatic paralysis of the newborn, the symptoms are not necessarily apparent immediately at birth. They generally come on within the first 24 hours. They may, however, not be present for some days. Upper extremity palsy of the Erb type is frequently present. It appears first and the dyspnea and cyanosis later. Approximately 80% of these paralytic cases recover completely.[26] This recovery in the majority of cases occurs within the first month,[14] but in some cases may take several months.[13,27] Of the remainder, a few do not survive. In those who do, the dyspnea and cyanosis disappear, but the diaphragmatic paralysis persists as in our Case 1, who had respiratory distress and cyanosis in the first few days of life, symptomatic recovery, but residual hemiplegia and partial diaphragmatic paralysis.

Pathological study has often shown muscle tissue in various degrees of degeneration in proven pathological cases. At times, the tissue appears normal on gross examination but shows degeneration microscopically. In the few cases of brachial plexus injury adequately studied at post-mortem there has been avulsion of the anterior roots of the plexus from the spinal cord.[12,26] The phrenic nerve has shown varying degrees of demyelinization.

One or two attempts to stimulate the phrenic nerve in the newborn period have been made, but the results are not impressive. If a contraction is not obtained on stimulation, it is assumed that the lesion is below the point of stimulus and vice versa.

In these cases, as in the cases classified as total eventration of the diaphragm, there is no way of making an accurate prognosis. Careful observation over the first few days of life will usually show whether the respiratory distress and cyanosis are beginning to clear or are remaining unchanged, and threatening the child's life. In the latter cases operative intervention appears to be indicated.

4. Eventration of the Diaphragm

It will be seen that in this class are left the lesions which are not clearly normal variations, are not diaphragmatic hernia in the usually accepted sense, and are not established diaphragmatic paralysis.[17]

Our Case 3 is the sixth such case to be successfully corrected by surgery when all cases reported as either diaphragmatic paralysis or total eventration of the diaphragm are included.

Pathologic examination sometimes makes differentiation of paralytic cases possible. In the paralyses, there may be avulsion of the brachial plexus, phrenic nerve demyelinization or degeneration, or diaphragmatic muscular degeneration. In the cases which can

be finally classified as total eventration clinically, radiologically and pathologically, the diaphragm is elevated in its entirety, the brachial plexus and phrenic nerve are intact (though the phrenic nerve may be smaller than average), and the diaphragm shows no muscular degeneration. It may show, as in our Case 7, absence of muscle tissue in the diaphragm or, in other cases, scattered areas of muscular fibres which are otherwise histologically normal.[5,11] A diaphragm which grossly appears fibrous but microscopically shows scattered muscle fragments has been quite frequently noted in previous reports.

All the six recorded cases successfully corrected surgically,[2,24] including our one, did not have associated brachial plexus injury or other neurological signs. There are many reports, however, of deaths in the cases clearly due to birth injury and phrenic nerve palsy. There seems no reason to believe that surgical correction of these cases could not be undertaken when necessary.

Partial Eventration of the Diaphragm

This is a sub-group of eventration. It is the least adequately reported in the literature, yet it forms the largest group in our cases. Although it is reportedly infrequent, compared with paralysis, total eventration and diaphragmatic hernia, it seems most likely that this is because it is usually asymptomatic and, therefore, does not attract attention.

Some of our cases, such as Case 6, the mother of two children with similar changes, must be considered as normal variants. Hers is the "antero-medial bulge". When it becomes more marked, as in her daughters, it may still be described as a normal variant or as a partial eventration of the diaphragm. Although our Case 4 was corrected surgically, she was asymptomatic before operation.

Case 7 was also asymptomatic, and operation was carried out because of the obvious abnormality of the left leaf of the diaphragm and possibly because of the previous episode of pneumonia for which he had been hospitalized elsewhere. Case 8 was discovered because of an episode of pneumonia. The lesion is now asymptomatic and presumably will continue to be so.

The partial eventrations are undoubtedly herniae. The sac, in these cases, appears to be fibrous or tendinous diaphragmatic tissue containing no muscle or only scattered muscle fibres. Whether occurring on the left or right side, it seems evident that this fibrous tissue does not permit a complete dislocation of abdominal contents into the thorax, as in the classical newborn diaphragmatic hernia, and that a progressive thinning or weakening does not occur during later life. They are, therefore, generally asymptomatic, though quite striking on X-ray evamination. One case is reported[28] of a female, age 47, who had a portion of the liver incarcerated in a diaphragmatic hernia which was apparently symptomatic for six months, and was operated on, with resulting relief of symptoms. In this case, the liver was intact and no resection of it was necessary. There are no other convincing reported cases of symptom-producing partial eventration of the diaphragm.

Diagnosis is usually not difficult. On the left side the abnormal diaphragm is generally outlined by the underlying gastro-intestinal gas. On the right side there may be more difficulty, but the lower edge of the liver is higher in position than normal. Fluoroscopy will show decreased movement of the affected segment of the diaphragm. It may or may not be "paradoxical", depending on the degree of thinness and weakness of the involved area. The periphery of the affected leaf will show normal movement synchronous with the opposite leaf.

In some cases, a localized eventration or hernia of the diaphragm, permitting a small area of the liver to bulge into the thorax, will closely simulate an intrathoracic tumour. There are several published reports of such cases. The authors have seen one case, but not at the Montreal Children's Hospital, and therefore not included in the present series. This was mistaken for an intrathoracic tumour until its true nature was revaled at operation. The avoidance of this diagnostic error involves remembering that any apparent intrathoracic tumour, inseparable from the diaphragm, may be a localized diaphragmatic eventration or hernia. In cases of doubt a diagnostic pneumoperitoneum will settle this point.[6] This has not been found necessary in any of the present series, and should, we believe, be reserved for those cases in which plain films and fluoroscopy do not establish the diagnosis with certainty. Pneumothorax has also been recommended as a diagnostic procedure,[10] but seems to us unsatisfactory and dangerous.

It is of considerable interest that diaphragmatic eventration, whether partial or total, in life never appears to be bilateral. The reason for this is not clear. The reported herniae and paralyses are also invariably unilateral. It becomes increasingly interesting and puzzling in view of our Case 11, who died following operation and showed a bilateral abnormality at post-mortem though the upward displacement was unilateral in life. This child also is a possible exception to the rule that partial eventrations are asymptomatic. Her whole hemidiaphragm on the affected

side was not involved at autopsy, yet she had severe cyanosis and dyspnoea. It seems reasonable to suppose that the diaphragmatic lesion was in the main responsible for the child's signs and symptoms, but that the congenital heart disease may have been a contributing factor.

Summary

Eleven patients with eventration of the diaphragm are presented. Three had total eventration; of these, two were paralytic lesions, associated with facial palsy and hemiplegia respectively.

Eight had partial eventration; of these, all but one were asymptomatic. Three had surgical correction. Two of these were asymptomatic pre- and post-operatively. One had severe cyanosis and dyspnoea pre-operatively, and died post-operatively, autopsy showing a complicating congenital heart lesion of non-cyanotic type which may have contributed to her illness and death.

The relation of eventration of the diaphragm to normal variations in the diaphragmatic contour, phrenico-diaphragmatic paralysis, and diaphragmatic hernia are discussed.

Total eventration of the diaphragm is likely to be symptomatic. Partial eventration is likely to be asymptomatic.

Acknowledgment

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CARCINOMA OF THE INFRAPAPILLARY PART OF THE DUODENUM

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The relative rarity of a pre-operative and/or pre-postmortem diagnosis of carcinoma of the infrapapillary portion of the duodenum is deemed as sufficient reason to present the observations and findings of yet another proven case, followed by successful surgery.

Prosen and Hastings-James[1] added one case to the 17 successfully resected cases previously reported by Poer[2], in 1953. Of these 17 cases Poer indicated that approximately one third were pre-operatively diagnosed. This percentage of pre-operative diagnoses is rather low even when one recalls the fact that carcinoma of the duodenum constitutes about 0.3% of all intestinal carcinoma[1].

Case Report

Mrs. P., age 69, reported to her doctor March 24, 1956, complaining of loss of weight, vomiting and anorexia gradually increasing over a period of 3 months. She was referred for X-ray examination on March 27, 1956. At this time she was thin and pale showing early signs of emaciation. There was slight fullness of the epigastrium but no mass was palpable. She had not experienced any pain.

The X-ray examination revealed subtotal obstruction at the infrapapillary region of the duodenum. There was too much food and fluid retention, despite 10-hour pre-examination starvation, to observe much mucosal detail; however, a tentative primary diagnosis of carcinoma of the duodenum was made at this time with a secondary consideration of carcinoma of the pancreas, (Figs. 1,2,). The stomach was moderately dilated and showed a 4-hour retention of 75%.

Figure 1. Right Lateral Projection. March 27, 1956.

Figure 2. Postero-anterior view. March 27, 1956.

On April 2, 1956, the patient was hospitalized; her physical condition on admission was unchanged. After gastric drainage, lavage and intravenous feedings for 3 days, she was re-examined. X-ray examination at that time provided a better opportunity for mucosal study, and Dr. Simor concurred in the primary diagnosis of carcinoma of the duodenum, his opinion being based on the marked localized constriction with proliferative mucosal changes proximal and distal to the constriction, as seen in Figure 3.

At surgery, the lesion was found to be localized to the duodenum with no sign of regional glandular involvement or extension to the pancreas but adherence to the pancreas was quite evident. Pancreatico-duodenectomy and resection of the stomach were performed.

Post-operatively, the patient developed a pancreatic fistula which cleared up by June 20, 1956. The patient also developed phlebitis which further complicated her recovery. She was discharged on May 23, 1956, and, at the time of reporting, July 16, 1956, she was at home, eating quite normally and gaining weight and strength.

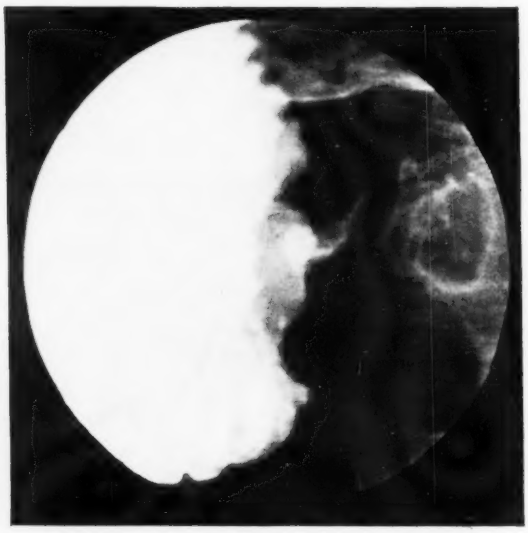

Figure 3. Spot-film on lesion. April 5, 1956.

The pathological diagnosis was adeno-carcinoma of the duodenum, sub-papillary region.

The questions that arise in this instance are — How much earlier could the radiological diagnosis have been made during the period of 3 months after symptoms first occurred and before the patient reported to her doctor? What better prognosis could have been anticipated, if earlier diagnosis had been possible and subsequent earlier surgery? Are we, as radiologists, routinely paying enough attention to parts of the gastro-intestinal tract beyond the duodenal cap?

Acknowledgment:

Our thanks to Dr. H. M. Wilensky for referring the patient and permitting this publication.

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URTICARIA PIGMENTOSA, WITH BONE LESIONS *
(Systemic Mast Cell Disease)

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Urticaria pigmentosa, until quite recently, has been known as a skin disease of benign, innocuous character, relatively self-limited and more common in childhood than in adult life; manifesting itself by discrete, brownish, pigmented macules or papules of the skin. A clinical characteristic is urtication after the lesions have been rubbed. The lesions, histologically, show an accumulation of tissue mast cells in the corium of the skin, and in dermal connective tissue. Although the disease is well represented in the dermatological literature, concomitant bone changes have not been recognized until recently. We have found, in the literature, only four reported cases of urticaria pigmentosa with skeletal changes of significance.

Case Report

We report here a fifth case of urticaria pigmentosa with bone lesions which we and our colleagues investigated in 1956. The patient was a 51 year-old white female, who presented a reddish-brown papular rash scattered all over the body. The patient stated that she had had a similar rash 20 years previously. At that time she was treated with "calcium injections" and the rash had disappeared for one year, but then re-appeared and had been present ever since. The spleen was definitely palpable, but the physical examination was otherwise normal. Her family history was non-contributory.

Urinalysis was negative.
Haemoglobin — 13.3 gms.
Hematocrit — 44.
Mean corpuscular haemoglobin content — 30.
Sedimentation rate — 6.
White blood count — 5000.
Platelets — abundant.
Prothrombin time — normal.
Prothrombin activity — 90%.
Clot retraction — 3+ (normal).

The haematologist considered that there was a significant shift of neutrophils to the left in the differential count. On bone marrow aspiration he demonstrated a striking increase in mast cells, somewhat less mature than those usually observed. Special blood-clotting studies before and after irritation of the skin lesions revealed no abnormality. A biopsy of the skin lesions, with special stain, demonstrated aggregations of tissue mast cells in the upper cutis. The pathological diagnosis was urticaria pigmentosa.

Radiological bone survey demonstrated a peculiar demineralization of the bones of the ribs, with coarsened trabeculation (Fig. 1). Several ribs showed evidence of old united fractures. The vertebral bodies (Figs. 2, 3, 4) of the thoracic and lumbar spine presented a similar coarsening of trabeculation, with an associated demineralization. There was slight loss of calcium density of the pelvis, and the skull showed slight demineralization of the inner table, with a slightly ground-glass appearance of the vault.

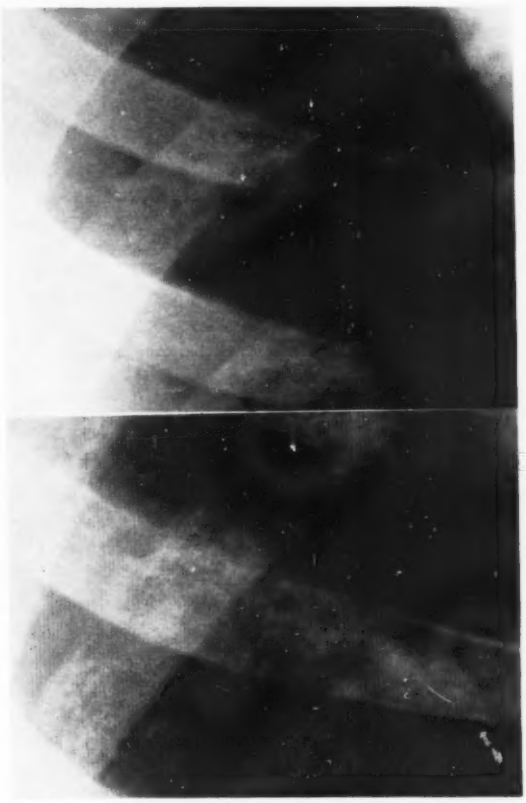

Figure 1. A. Demonstrates demineralization of ribs, with small radiolucent areas in ribs. No expansion of bone. Note coarsened trabeculae.
B. Similar radiograph of normal patient for comparison.

Previously reported cases

The four other cases of urticaria pigmentosa with bone lesions which have been reported in the literature, include the one reported in 1952 by Felix Sagher, Cohen and Schorr.[16] That patient had bone changes which suggested, radiologically, a generalized demineralization of the ribs, vertebrae and skull, which they described as "multicystic." Unfortunately, they were unable to obtain a biopsy specimen.

*Presented at Annual Meeting, The Canadian Association of Radiologists, January 13-17, 1957, Montreal.

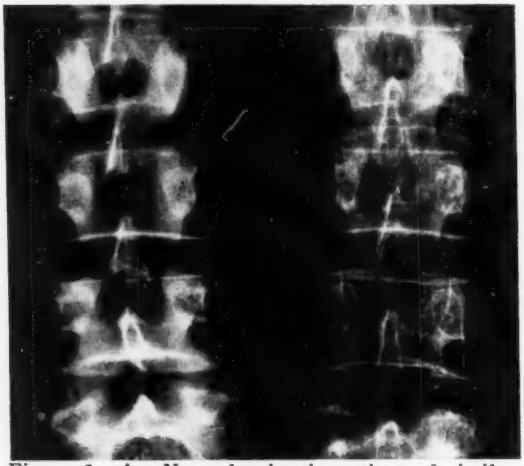

Figure 2. A. Normal spine in patient of similar age.
B. Note marked demineralization, with very coarse trabeculae present. Again there is no expansion of bone.

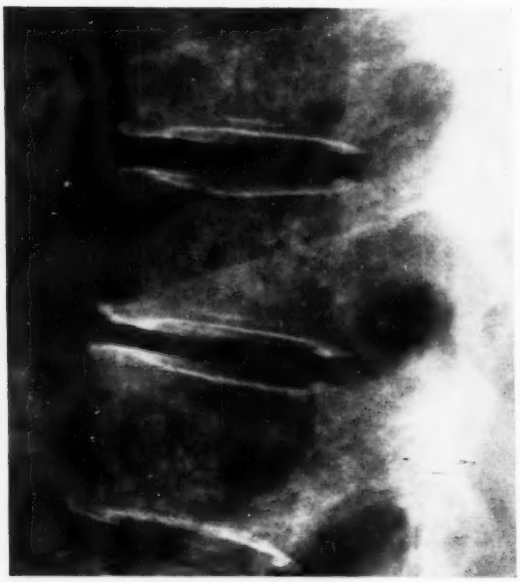

Figure 3. Lateral view of the lower thoracic vertebrae showing coarsening of trabeculation and loss of mineral content.

Sagher and his associates assumed that urticaria pigmentosa might well be a systemic disease, and that the bone lesions their patient presented might be due to mast cell infiltration of the bone marrow.

In October of 1956, Sagher[13] reported further on the same patient, and added another (the fourth) case — a 55 year-old female who had definite clinical urticaria pigmentosa, with radiological evidence of a similar "multicystic" osteoporosis of the ribs and vertebrae. The skull was also involved. She died

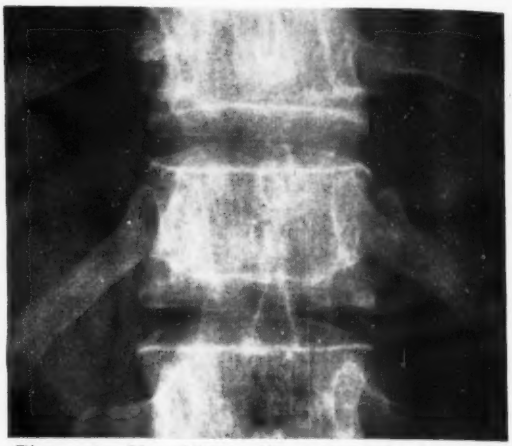

Figure 4. Magnified radiograph of the lower thoracic vertebrae, again demonstrating the very coarse trabeculation.

of monocytic leukemia. At autopsy, the bones showed, histologically, dense accumulations of tissue mast cells in the diploe of the skull and in most of the marrow spaces of the other bones. Neither of Sagher's two cases had any symptoms or signs of bone pain or disability from bone involvement.

Ellis,[7] in 1949, had reported the autopsy of a child, one year old, who had typical urticaria pigmentosa. This patient had tissue mast cells in the skin lesions, in the bone marrow, the spleen, liver, kidney and pancreas.

In 1952, Clyman and Rein[5] reported 8 cases of urticaria pigmentosa, none of whom on bone survey demonstrated the bone changes seen in Sagher's cases (one had multiple hereditary osteochondromas, and another had multiple small bone infarcts in the long bones — femora and humeri).

In 1953, Asboe-Hanson[1] described a 13 year-old boy who had bilateral fragmentation of the epiphysis of the olecranon and urticaria pigmentosa. He was skeptical that these bone changes had any relation to the skin disease, but stated he was awaiting with interest a case report with bone biopsy.

Bluefarb,[3] in 1954, reported the case of a 30 year-old man who had urticaria pigmentosa; marked dermographism (which may be a feature); severe gastro-intestinal symptoms; splenomegaly and, radiologically, a generalized "cystic osteoporosis" of the ribs, with some coarsening and thickening of trabeculae. The thoracic and lumbar vertebrae showed the same changes. Unfortunately, no bone biopsy was obtained. He considered, further, that urticaria pigmentosa could no longer be regarded solely as a skin disease, but as a systemic disease in view of the increasing number of reports of bone and gastro-intestinal involvement.

Reilly, Shintani and Goodman,[14] with a review of the literature, in 1955 added another case of urticaria pigmentosa with systemic involvement. Their patient was a 34 year-old man with gastro-enteritis, typical proven urticaria pigmentosa, bone lesions (as in Sagher's cases), and hepato- and splenomegaly. Bone aspirates from ribs and sternum disclosed aggregations of tissue mast cells. A liver biopsy revealed tissue mast cells.

Sagher *et al*[13] surveyed 8 adults (including the two noted above) and 7 children with urticaria pigmentosa. Among these 15 cases, only those two had generalized lesions of the bones such as we observed in our case. In his patients the skeletal involvement had been known to be present for 5 years in the one case, and for 2 years in the other, without any clinical symptoms referable to the bones.

Discussion

The bone lesions present in our case are, of course, not pathognomonic of urticaria pigmentosa. The main problem was in the differentiation of this disease from multiple myeloma. As can be seen, the textural changes in the ribs, particularly as to local rarefaction, might be difficult to differentiate from those of early multiple myeloma, but in the full-blown picture would not present this difficulty.

Sagher considers that osteitis fibrosa deformans is the major disease to be differentiated from mast cell involvement, on the basis of the coarsened, osteosclerotic appearance of the increased trabeculation. We do not perceive the fibrillar or lamellated proliferation of bone which is characteristic of Paget's disease, and hence differ from his opinion. We believe that the demineralization and coarsened trabeculation is the reaction of the bone to the accumulation of abnormal tissue mast cells in the marrow.

Tissue mast cells occur in all regions of the body and are intimately associated with the connective tissue. Bloom[2] believes that they are not of haemic origin but are derived from mesenchymal cells. Moderate numbers of these cells are present in the superficial and deep fascia, and about the hair follicles. The odd mast cell is found in the bone marrow of mammals. These mast cells tend to align themselves along very small blood vessels and capillaries.

Tissue mast cells contain cytoplasmic granules which can be seen under suitable staining methods. Investigators have shown that the mast cell holds a mucopolysaccharide which is closely related to heparin, hyaluronic acid, and another substance, histamine. The granules in the tissue mast cells are thought by some authors (notably Frank Bloom, Jean Oliver and Mangieri)[11] to be precursors of heparin. Bloom has described mast cell tumours in dogs. These are not uncommon, and if the tumours are of a mature type, the heparin content of the tumour may be 200 to 300 times that of normal tissue in the dog. Bloom also states that dogs will develop a malignant type of mast cell tumour which metastasizes.

The fact that mast cells do produce heparin, or a heparin-like substance, would cause one to think that the blood coagulability of patients with urticaria pigmentosa would be altered. This, however, has not been so in the cases reviewed here, nor in our own case, for an increase of heparin has not been observed in the coagulation pattern of patients with urticaria pigmentosa. Perhaps the heparin is protein-bound, or the methods employed are not adequate to measure such an increase.

If the mast cell does produce histamine, or a histamine-like substance, the linkage of the mast cell infiltration of the skin and the urtication of the lesions becomes more logical and understandable.

Brodeur and Gardner[4] suggest that urticaria pigmentosa is a congenital metabolic error of histamine and heparin, and that to this the mast cell proliferation is secondary.

Summary

A patient with urticaria pigmentosa and associated bone lesions has been described.

Up to 1952, urticaria pigmentosa had been considered to be a benign, self-limited skin disease, causing only minor discomfort and with no associated systemic effects. Evidence has accumulated, however, that this is a systemic disease in an appreciable number of patients, manifesting visceral involvement in some — skeletal changes in others.

The case reported here brings to five the number of known cases with definite skeletal alteration.

Radiological differential diagnosis must consider it in comparison with multiple myeloma. We do not believe that its manifestations should create major confusion with osteitis fibrosa deformans. The bone lesions are undoubtedly associated with the aggregates of tissue mast cells in the bone marrow.

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Recently in the New England Journal of Medicine (January 10, 1957), F. G. Zak, J. A. Covey and J. J. Snodgrass reviewed the literature in urticaria pigmentosa. They found 21 cases reported. They do not divide the cases into systemic types and localized lesions which may or may not be due to urticaria pigmentosa.

MEETINGS

The Canadian Association of Radiologists — June 1957

The Special General Meeting of the Canadian Association of Radiologists is to be held at The Macdonald Hotel, Edmonton, as follows:

Monday, June 17, 1957 Meeting of Council 9 A.M. — 5 P.M.

Tuesday, June 18, 1957 General Meeting 7 P.M. (Dinner meeting.)

Reduced rail travel: The Canadian Medical Association has sent a supply of identification vouchers for reduced *rail* travel. Any member wishing to obtain such a voucher may do so by applying to the Central Office of the C.A.R.

The Canadian Association of Radiologists — January 1958

The Twenty-First Annual Meeting will be held at the Hotel London, London, Ontario, as follows:

Sunday, January 12, 1958	Meeting of Council
Monday, January 13, 1958	Meeting of Council
Tuesday, January 14, 1958	Annual General Meeting Scientific Sessions
Wednesday, January 15, 1958	Scientific Sessions

MEDULLARY LIPOMA OF BONE *

G. BERNARD SKINNER, M.D. and ROBERT G. FRASER, M.D.
Montreal, Quebec

Fatty tissue occurring as a normal constituent of the marrow cavity of adult long bones is well recognized. However, the possibility of the presence of fatty tissue as a form of benign neoplasm in this location is not commonly realized; in fact, benign lipoma is not included in the classification of bone tumours in three standard texts on this subject.[6,9,10] Excluding generalized lipomatosis, in which bone is involved only by pressure from adjacent soft tissue lipomas, there exist two sites of origin of the primary form of these tumours — the subperiosteal area, and the medullary cavity. The periosteal type of lipoma is an uncommon but not rare lesion. The medullary variety, on the other hand, is indeed rare, there having been only four previously reported cases. The discovery of such a tumour recently in a patient in the Royal Victoria Hospital has given us an opportunity to bring the existence of such a lesion to your attention and to attempt to establish criteria for its diagnosis.

This 56 year-old male was in good health until 8 weeks before his admission to hospital at which time a fall from a train resulted in a constant throbbing pain in the region of his left hip. One week after the accident he first noticed pain and tenderness in his left shoulder, which he did not attribute to the accident. The shoulder pain had a sharp, jabbing quality but did not radiate. An area of tenderness could be localized to the upper region of the left arm, underlying the shoulder muscles. This pain gradually changed to a dull constant ache, with intermittent episodes of sharp stabbing pain when the arm was abducted or rotated. It gradually diminished, only to recur in its original severity 2 days before admission. He never had symptoms referable to other joints in his body, except for a so-called "neuritis" in the left shoulder region 25 years previously, during the cold winter months.

On admission, clinical and radiographic examination of the left hip did not reveal any significant abnormality.

The left shoulder showed limitation of full abduction and rotation because of a sharp jabbing pain localized to the same area. There was no decrease in deltoid tone nor in muscle power of the left shoulder compared to the right.

Radiographic examination of the left shoulder included plain films Fig. 1 and tomographic studies Figs. 2 and 3 in antero-posterior projection, and demonstrated a

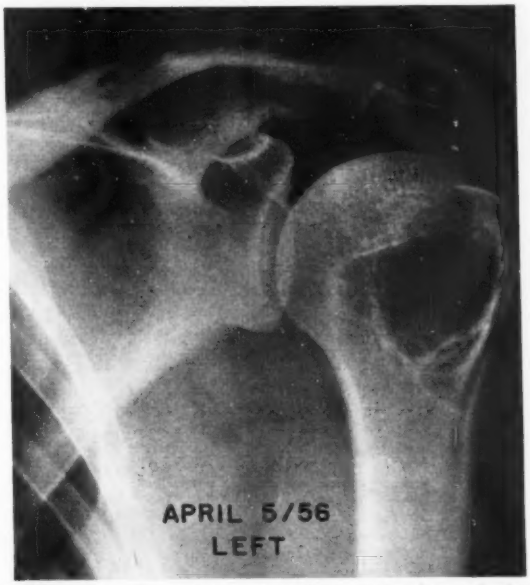

Figure 1. An antero-posterior projection of the left shoulder showing a sharply defined cystic lesion in the head of the left humerus.

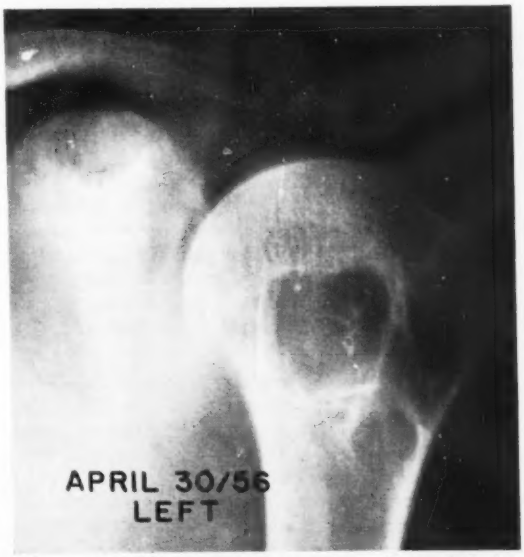

Figure 2. A tomographic section of the left humerus demonstrating the well corticated margin and bony septae that divide the lesion into several compartments.

Presented at Annual Meeting, The Canadian Association of Radiologists, January 13-17, 1957, Montreal.

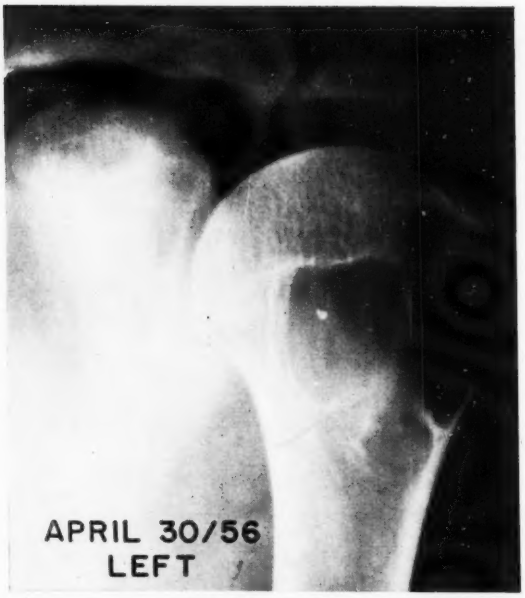

Figure 3. A tomographic section, 2 cm. posterior to Fig. 2, again demonstrating the bony septae, and corticated margin of the lesion, with normal surrounding bone.
Note: The white spot in the centre of the lesion is an artefact.

sharply defined cystic lesion in the proximal end of the left humerus, localized chiefly to the metaphyseal area but showing minimal extension across the old epiphyseal line. The lesion measured 4 x 3 cm. in diameter and possessed a thin, dense shell of cortical bone around all margins. The surrounding bone was of normal density and trabeculation, and the cortex was not eroded. There was no expansion of the regional bone, and no subperiosteal new bone formation. A few coarse septae of bone divided the lesion into several compartments of varying size. It was felt necessary to include in the radiological differential diagnosis all the various conditions which were known to cause cystic areas of rarefaction in the metaphyseal region of a long bone, the most likely of which were considered to be: (a) fibrous dysplasia, (b) a simple bone cyst in an atypical site and (c) a giant cell tumour.

The patient proceeded to operation and on exposure of the upper end of the humerus, the periosteum and underlying cortex were found to be intact and normal in appearance. Immediately upon cutting through the cortex, a soft yellow tumour was encountered. The area was curetted and it was noted that the operative cavity contained tiny oil droplets. After thorough cleansing, the cavity was filled with bone chips and the wound closed. The patient made an uneventful recovery.

On pathological examination, the gross specimen consisted of several small glistening yellowish fragments of tissue with a distinctly greasy appearance, the largest fragment measuring 1 cm. in diameter. Scattered through these small pieces of tissue were spicules of spongy bone. Other portions of the specimen were made up of greyish-white and yellowish tissue which was partly haemorrhagic. Microscopic examination of serial sections through all the material submitted revealed areas of haemorrhagic mature fatty tissue, including foci of myeloid elements. There were also fragments of mature and viable bony trabeculae. Several of the sections showed radiating cleft-like spaces surrounded and separated by proliferative connective tissue, in which were distributed mononuclear cells and multinucleated giant cells of foreign-body type. Epithelial elements were completely lacking, and there was no lining membrane. The histological appearance was that of a vascularized mature fatty tissue, and the pathological diagnosis of Lipoma of Bone, of Intra-osseous Variety, was made.

Discussion

Review of the literature reveals that four previous cases of intra-osseous medullary lipoma have been recorded.[5,7] The fact that these lesions occurred in the femur, the tibia, the fibula, the calcaneus and, in the present case, in the humerus, indicates the lack of predilection for any specific bone. In addition, the tumours have been found in both sexes, of an age group extending from 5 to 56 years. Although it is realized that no statistical significance can be derived from a series of only 5 cases, there exist certain radiological features common to all cases thus far reported which, as a group, seem to be sufficiently distinctive to enable one to suggest the possibility of this entity in the differential diagnosis of any cystic lesion of long bones. The features referred to are five in number, namely:

1. A constant relationship to the medullary portion of the end of a bone.
2. The presence of a well-corticated margin surrounding the tumour.
3. The lack of any tendency toward erosion of the enclosing cortex.
4. The absence of any stimulus on the part of the tumour to produce subperiosteal new bone.
5. The presence of incomplete coarse bony septae dividing the tumour into compartments of varying size.

When these five criteria are fulfilled in any case of a characteristically benign lesion of bone, *especially* in a patient in the older age group, we believe that the possibility of intramedullary lipoma may be strongly entertained. However, it must be emphasized that histological examination of biopsy material is essential before a definitive diagnosis can be made.

Confusion has existed in the past between the medullary variety of lipoma and its periosteal counterpart. The distinction lies in the fact that the latter form is intimately associated with the periosteum, and exerts its effect on the bone by pressure erosion of the cortex from without. An even more common manifestation is its tendency to extend in an outward direction and to create a soft tissue mass, thereby allowing obvious differentiation from the intra-osseous variety.

Summary

1. The discovery of a benign medullary lipoma in the humerus of a 56 year-old male patient in the Royal Victoria Hospital, brings the number of reported cases of this tumour to five.

2. Radiologically, five features have been found to be present in the majority of these cases:

(i) A constant relationship to the medullary portion of the end of a bone.

(ii) The presence of a well-corticated margin surrounding the tumour.

(iii) The lack of any tendency toward erosion of the enclosing cortex.

(iv) The absence of any stimulus on the part of the tumour to produce subperiosteal new bone.

(v) The presence of incomplete coarse bony septae dividing the tumour into compartments of varying size.

3. It is not suggested that these features are diagnostic in any one case, but that they comprise a group of criteria upon which the diagnosis may be strongly suspected, especially in a patient in the older age group. As in other bone tumours, a definite diagnosis rests with histological examination of biopsy material from the tumour site.

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